portance in the lives of physicians, has passed. Today, it behooves every member of the medical profession to be fully alert, and take an active interest in organization work; because, only through united effort will it be possible for us to preserve those methods which experience has shown to be necessary implements, in making possible the highest type of medical service for the citizens of our state and nation. I thank you.

TRAUMATIC RUPTURE OF THE SPLEEN*

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AND
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DISCUSSION *by Alanson Weeks, M. D., San Francisco; F. Paul O'Hara, M. D., San Diego.*

THIS paper is a review of twenty-one cases of injury to the spleen. Although there are many papers appearing in the literature discussing this relatively uncommon injury, it deserves renewed attention because (1) there are not a few autopsies revealing unrecognized ruptured spleen as the major cause of death, and (2) surgeons not dealing primarily with trauma fail to allot this subject its proper importance. We must constantly keep in mind the possibilities of rupture of the spleen, rupture of the urinary bladder, and rupture of other hollow viscera in every injured person.

In this series there were seventeen males and four females, whose ages averaged twenty-three years. This predominance of younger males is undoubtedly due to the fact that they are most exposed to the types of trauma necessary to cause this injury. Thirteen of these resulted from vehicular accidents, five from penetrating wounds, and three from falls.

The interval between the time of injury and entry into the hospital varied from ten minutes to twenty-four hours, while the period of observation was from fifty minutes to fifty-five hours. These intervals were prolonged in some cases because of (1) the delay by the patient in seeking medical care, (2) the time consumed in an attempt to combat shock, (3) the time required for observation and the difficulty in making the diagnosis in the presence of multiple injuries, and (4) the fact that the ruptured spleen was overlooked because of other injuries.

DIFFICULTIES IN DIAGNOSIS

The difficulties in making a diagnosis may be due to (1) the frequent absence of visible signs of injury to the anterior abdominal wall which leads the patient, and, at times, the doctor, to disregard the early mild symptoms, believing any trauma sufficient to rupture the spleen would cause visible evidence of injury to the superficial tissues; (2) the characteristic recession of symptoms, as mentioned below; (3) the presence of shock resulting from head injuries, or alcoholism interfering with the usual reaction of the patient to injury; and (4) the presence of more obvious symptoms resulting from other injury.

PAIN

Regional pain is the most important clinical manifestation of rupture of the spleen. It is often misleading in character. Pain may be of a dull, aching type, or it may be sharp and lancinating. It may be localized to left upper quadrant early, and soon generalized throughout the abdomen. Generalization occurs as the blood spreads in the peritoneal cavity. The pain is usually more intense in the left upper quadrant or left flank. Characteristically, the pain continues during active bleeding. This recurrence of pain, due to recurring hemorrhage, is partly due to the rhythmic contractions of the spleen, which gives the following cycle: A clot forms, the patient's condition improves, blood pressure rises, contraction of the viscus dislodges the thrombus, bleeding recurs, and pain and shock once more become evident. The pain is not severe, as a rule. It does not reach the intensity of that in rupture of the stomach or duodenum. It is usually relieved by small doses of opiates. It is similar to that present in the lower abdomen following a rupture of an ectopic pregnancy.

At times, radiation of pain to the shoulders and left back occurs because of irritation of the diaphragmatic peritoneum, and attention may be erroneously attracted to the chest.

REËXAMINATIONS IMPORTANT

The injury often is not suspected in the patient who is most helped by prompt surgical intervention. Hourly examination and rechecking of the findings and laboratory work in all traumatized individuals are essential. Often by the time the resident examines the patient who has been reported in shock, the bleeding has temporarily ceased and general condition has improved, thus misleading the examiner.

In all patients where there is a history of trauma to the abdomen, the flanks or the lower chest, even if there be no visible injury and no early complaint of pain, we must be spleen-minded and not overlook the slowly developing shock, the slight distention, the mild abdominal pain and slight discomfort, which are the early signs of rupture of the spleen.

Tenderness, direct and rebound, and guarding coincide with the distribution of the pain, but they may not be remarkable. Freed blood in the peritoneal cavity is seldom demonstrable, even when a considerable quantity is present, although localized dullness may indicate a confined hemorrhage in the splenic area. This local collection of coagulated blood is sometimes visible on the roentgenogram. Rectal tenderness and a soft, resisting fullness may be present due to blood in the cul-de-sac of Douglass. Temperature, pulse, respiration, and blood pressure usually are indicative to some degree of shock. The temperature is subnormal, the pulse may be elevated to 120 or more per minute. A fact, not well known, is that early in hemorrhage the

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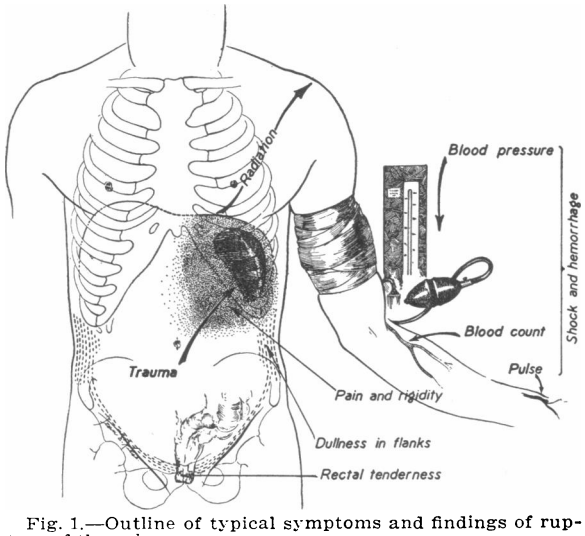

Fig. 1.—Outline of typical symptoms and findings of rupture of the spleen.

pulse slows in an attempt to encourage coagulation to lessen the amount of blood loss. The blood pressure fluctuates with the hemorrhage. The red blood cell count and percentage of hemoglobin often do not point to severe hemorrhage, but a slight gradual fall is significant. The peripheral count (ear, finger) often is deceptive because of the peripheral concentration and stasis of the blood in shock. Blood from the larger veins (the so-called "central count") is of more value. The white blood count is usually high. All but four of this series showed more than 20,000, and one 41,000 white blood cells. The polymorphonuclear count is, likewise, high. Only four in this series fell below 80 per cent, and one showed 98 per cent.

ASSOCIATED INJURIES

The majority of these patients, especially those with penetrating wounds, had severe associated injuries. In those with nonperforating injuries, five were associated with fractured skull, ribs, pelvis, femur, and forearm. One had a ruptured duodenum, one a ruptured liver, and one a ruptured kidney. It may again be emphasized that these injuries are apt to confuse the picture and draw attention to the obvious injuries, causing the symptoms of splenic injury to be overlooked.

It should not be forgotten that the spleen is within the rib-cage as well as being intra-abdominal—as are parts of the stomach, liver, and large bowel—and may be injured in penetrations of the chest. In only three of our records was any mention made of evidence of external injury in the subcutaneous splenic rupture. There may be abrasions or ecchymosis at the site of injury, but the latter, especially, is not evident at the time of early examination.

The treatment of a ruptured spleen is splenectomy. If the spleen is comminuted or completely torn from its pedicle, immediate intervention is imperative. A few hours may be disastrous. Death from exsanguination has taken place in such patients within a few hours. Ordinarily, in severe shock, it is rational not to operate upon the patient, but treat the shock first. However, if the shock is due primarily to hemorrhage from large vessels, then immediate surgery is indicated. Administration of intravenous fluids and transfusion should be reserved for the operating room or postoperative treatment. Usual quantities of intravenous fluids may so raise the blood pressure that exsanguination rapidly takes place.

EXPLORATION OF ABDOMEN

When exploring the abdomen, complete relaxation and ample incision are essential. Ether or subarachnoid block are the anesthesias of choice. The spleen must be carefully palpated. This is particularly true of palpation in the region of the junction of the pedicle and hilus posteriorly, because it is here that important injuries may escape the exploring hand. If free, uncontaminated blood is encountered it may be dipped out with a small ladle or aspirated, citrated and autotransfused. If not sufficient blood is recovered to dilute the citrate solution, normal saline may be added. Autotransfusion is at times a life-saving procedure. At operation, the spleen has been found to be ruptured in various degrees. No doubt, there are superficial tears giving few symptoms and few signs of peritoneal irritation, not often diagnosed as splenic injury, which recover rapidly with rest. The severe lacerations usually run transversely or obliquely to the long axis.

MORTALITY

The mortality in our group of patients was as follows:

Number of cases	21
Died	8
Mortality	38%
Nonpenetrating injuries	16
Died	5
Mortality	31%

Of the five that died, one also had a rupture of the duodenum; one had a ruptured spleen, which was not found at operation; two died within forty-eight hours of shock and hemorrhage; one died within ten days of peritonitis and pneumonia.

CONCLUSIONS

1. Traumatic rupture of the spleen is often not found until autopsy.
2. Many factors contribute to the frequent delays in surgical intervention.
3. Diagnosis may be difficult, although this injury presents a fairly typical course of clinical events (Fig. 1).
4. The prognosis is grave and the mortality high.
5. Splenectomy is the treatment.

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DISCUSSION

ALANSON WEEKS, M. D. (384 Post Street, San Francisco).—Doctors Butler and Birnbaum's paper, presenting twenty-one cases, is certainly well worth careful consideration. The fact that ruptures of the spleen are often first found at autopsy indicates that this statement of the doctors is not analyzed as to differentiating those cases with death from splenic hemorrhage alone from those ending from associated injuries in which splenic hemorrhage was a contributory factor. Certainly, one cannot deny the need of careful examination of injured patients with the diagnosis of splenic hemorrhage in mind. In our experience the

patient does not, as a rule, show external marks which would lead one to suspect an injury of the spleen. The diagnosis is made clinically. Occasionally an injury that is apparently trivial can cause such a hemorrhage. We recall one schoolboy who jumped on a street-car. While holding an exercise book with one edge in the fold of the left elbow and the other against the flank, he received a bump on the elbow as he swung onto the car. This was sufficient to rupture the spleen. Another case, a small girl, fell off a garage roof; she received bilateral Colles's fractures and a ruptured spleen. One must, therefore, constantly keep the possibility of a ruptured spleen well in mind. When in doubt, explore. The dangers incident to a simple exploratory operation, with negative findings, are far less than the danger of overlooking some lesion, the neglect of which may be fatal.

F. PAUL O'HARA, M. D. (Medico-Dental Building. San Diego).—This timely article on the very interesting subject is of an especial appeal to me, since, upon reviewing the cases of ruptured spleen in this district two years ago, I found that only one-third of the cases were diagnosed and operated upon prior to autopsy.

The difficulty of diagnosis is emphasized by the authors, and usually only a diagnosis of some ruptured viscus in the abdomen is possible. The initial shock in a ruptured spleen of any severity is out of proportion to the findings and should make one suspect the pathology. In cases that are observed longer than twenty-four hours following an injury, and in which the initial shock is overcome, the majority, if not all the cases, show a rather marked rise in blood pressure above normal, even in young people, which persists unless severe hemorrhage occurs, and lasts as long as several months following splenectomy. This interesting phenomena has not been satisfactorily explained by any investigators.

In some of the cases that I have seen, it is remarkable that the patient has gone back to his home for a period of a week or so following injury without a diagnosis being made, when the release of a blood-clot with resultant hemorrhage causes him to return with a typical picture. Another diagnostic point, which is not present, however, in all cases, is hematemesis of old blood, which possibly may occur due to the close association of the blood supply of the spleen and the stomach. The emphasis of close, continued reëxamination of these cases by the authors is to be commended, and the prompt, proper carrying out of splenectomy results in complete recovery in a surprising majority of cases. The use of autotransfusion is sometimes a life-saving procedure, but in all events transfusion or infusions should be saved until the hemorrhage is stopped by the performance of splenectomy.

IS PARESIS (DEMENTIA PARALYTICA) CURABLE?*

RESULTS IN PERSONALLY TREATED CASES

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DISCUSSION *by Samuel D. Ingham, M. D., Los Angeles; Benjamin W. Black, M. D., Oakland.*

THE clinical terms, general or progressive paresis of the insane, or dementia paralytica—paresis, in common parlance—given us by the older writers, describe only the final stages of this disorder, as seen in institutions and neglected cases. These terms are still applied to the earlier stages of the disease, when physical weakness or advanced mental disintegration is not evident. Our knowledge of paresis has outgrown the nomenclature, and there is need of a more descriptive term having a precise clinical and pathological foundation; however, the term is well established by usage and no descriptive terminology has satisfactorily replaced it. The disease is a pathological entity and is one of the few neuropathological conditions which may be microscopically diagnosed. To the classical description of Alzheimer [1] only a few additions have been contributed in recent years, notably the presence of the Spirochaeta in brain tissues and the characteristic iron deposits in the adventitia of the blood vessels. He had already (1904) expressed the opinion that rod cells, now classified as microglia, were of mesoblastic origin.

The usual paretic brain observed at necropsy shows a predilection of the pathologic process for the anterior parts of the brain, and thickened and adherent membranes over the shrunken cortex. Microscopically, both chronic inflammatory and degenerative cortical lesions are present. The relationship of these associated lesions has never been satisfactorily explained. Alzheimer has discussed mental symptoms in syphilitic brain lesions and classifies the nonparetic forms as vascular, gummatous, and meningiomyelitic (parenchymatous). In the latter form he particularly calls attention to the important association of the syphilitic meningitis with the essential brain changes. This writer discards the meningeal theory of paresis on the basis that in the early stages of the disease the superficial cortical lesions may be so slight and lesions in the deeper cortical layers so marked, that a causal relationship is not to be reasonably entertained. As these meningeal changes are not only hyperplastic, but also infiltrative, the reservation is made, however, that they may exert a determining influence on the manifestations of the disease.

The lack of pleocytosis in the cerebrospinal fluid, in cases where necropsy shows markedly thickened cerebral membranes, suggests that these membranes were primarily the seat of an active meningitis, and later have become sclerosed, when the active process has been arrested. If this hypothesis is valid, an appreciable pleocytosis is to be expected in the first stages of the disease, and not necessarily in the later stages. Such a condition is comparable to pathological processes observed in tabes.

A high fluid pleocytosis, associated with a paretic curve and strongly positive syphilitic serology, is frequently met with in patients with symptoms of emotionalism, nervousness, tremor, and heightened reflexes, who are often referred to a neuropsychiatrist with a Wassermann-fast blood. Symptomatology does not disturb the patient so much as consciousness of persisting syphilis. Indeed, it is quite probable that such cases, because of disregard of symptoms, often remain undiagnosed and untreated, until some definite mental or moral aberration enforces medical observation and care. Whether such early cases show a characteristic pathology of paresis is unknown, as only a chance necropsy could definitely decide this question.

If acute syphilitic meningitis affects the base of the brain, cranial nerves are involved; if it affects the cortex, lancinating headaches, tendency to somnolency, hypo-activity, and symptomatic complaints of the patient, differentiate this form from

* Read before the Neuropsychiatry Section of the California Medical Association at the sixty-sixth annual session, Del Monte, May 2–6, 1937.